MEDICAL PROCEEDINGS

MEDIESE BYDRAES

A South African Journal for the Advancement of Medical Science

'n Suid-Afrikaanse Tydskrif vir die Bevordering van die Geneeskunde

P.O. Box 1010 · Johannesburg | Posbus 1010 · Johannesburg

Editor : Redakteur

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Vol. 7 | 9 September 1961 | No. 18

REDAKSIONEEL · EDITORIAL

PERSONE WAT BY 'N LYKSKOUING AANWESIG MAG WEES

In die geval van lykskouings op lyke waar die dood aan natuurlike oorsake te wyte was, word geen besondere moeilikhede teëgekom deur mediese praktisyns wat belang daarby het om die lykskouing by te woon nie. Wanneer die lyk van 'n persoon wat nie ten gevolge van natuurlike oorsake oorlede is nie egter ondersoek word, word sekere beperkinge deur artikel 3 (5) van die Wet op Geregtelike Doodsondersoeke, Nr. 58 van 1959, neergelê:

(5) By 'n ondersoek wat deur 'n geneesheer ingevolge sub-artikel (2) uitgevoer word, mag niemand behalwe—

(*a*) 'n polisiebeampte; of

(*b*) 'n ander geneesheer aangewys deur iemand wat die landdros binne wie se regsgebied daardie ondersoek plaasvind, oortuig dat hy 'n wesenlike en besondere belang in die uitslag van die ondersoek het,

aanwesig wees nie sonder die toestemming van daardie landdros of die geneesheer wat die ondersoek uitvoer.

Dit is 'n nuwe bepaling wat op 1 Januarie 1960 in werking getree het. Die doel daarvan is om die omstandighede te bepaal waaronder sekere persone lykskouings, uitgevoer kragtens die Wet op Geregtelike Doodsondersoeke, kan bywoon. Dit is doodsondersoeke waar die outopsie-bevindings nie verder as 'n formele lykskouing gevoer word nie; maar in sommige gevalle kan hulle die grondslag van kriminele vervolging of siviele gedingvoering word.

Persone sonder behoorlike magtiging word deur die statutêre bepalings verhinder om hierdie outopsies by te woon. In alle omstandighede, maar met uitsluiting van polisiebeamptes, kan òf die betrokke magistraat òf die mediese praktisyn wat die ondersoek waarneem (by die

PERSONS WHO MAY BE PRESENT AT A POST-MORTEM EXAMINATION

In the case of autopsies on bodies where death has been due to natural causes, no particular difficulty is encountered by medical practitioners who have an interest in being present at the examination. When, however, the examination is being conducted on a body dead from other than natural causes, certain restrictions are laid down in terms of sec. 3 (5) of the Inquests Act, No. 58 of 1959, viz.:

'(5) At any examination conducted by a medical practitioner in terms of sub-section (2) no person other than—

(*a*) a policeman, or

(*b*) any other medical practitioner nominated by any person who satisfies the magistrate within whose area of jurisdiction such examination takes place, that he has a substantial and peculiar interest in the issue of the examination,

shall be present without the consent of such magistrate or the medical practitioner conducting the examination.'

This is a new provision which came into effect on 1 January 1960. Its purpose was to regularize the conditions under which certain persons could be present at autopsies held in terms of the Inquests Act. These are post-mortem examinations in which the autopsy findings may not be pursued beyond a formal inquest, but in some cases they may become the basis of a criminal prosecution or civil litigation.

The statutory provision excludes persons from being present at these autopsies without proper authority. In all circumstances, but with the exclusion of policemen, either the magistrate concerned or the medical practitioner conducting the examination may (in the exercise of a general discretion) give the

uitoefening van hul algemene diskresie) die nodige verlof toestaan. Aangesien geeneen van hierdie beamptes oor meer gesag as die ander beskik nie, is daar 'n teoretiese moontlikheid dat 'n konflik tussen die magistraat en die mediese praktisyn kan ontstaan.

Afgesien van die verlof wat toegestaan kan word deur die magistraat of die mediese praktisyn, elkeen van wie na goeddunke handel, word 'n reg ook aan sekere persone verleen om teenwoordig te wees, en hierdie reg is nie afhanklik van die diskresie van die betrokke magistraat of die mediese praktisyn nie. Die 2 klasse wat van die algemene bepaling uitgesonder word, is:

(*a*) Polisiebeamptes.

(*b*) Mediese praktisyns, aangewys deur persone wat 'n wesenlike of besondere belang by die resultaat van die ondersoek het, mits hulle die magistraat tevrede kan stel dat hulle wel so 'n belang het.

'n Sekere mate van onduidelikheid word deur die gebruik van die woord ‚aangewys' geskep. Dit is, byvoorbeeld, nie duidelik of, indien die persoon wat 'n wesenlike of besondere belang het 'n mediese praktisyn is, hy homself kan aanwys nie. Die bepaling lê baie beslis neer dat gewone burgers in die algemeen nie teenwoordig kan wees nie. Dit lyk dus asof die blote feit dat die betrokke burger toevallig 'n mediese praktisyn is, geen regte aan hom toestaan wat groter as die regte van ander burgers is nie. As hierdie vertolking korrek is, beteken dit dat so 'n praktisyn 'n ander praktisyn sal moet benoem om hom te verteenwoordig. Afgesien van die inherente korrektheid van hierdie prosedure is dit ook wenslik om ander redes.

As ondersoek ingestel word na die oorsaak van dood waar daar onwettige gedrag was, kan die mediese praktisyn bes moontlik die verantwoordelike persoon wees, bv. in die geval van dood geassosieer met anestesie, 'n aborsie, moord, ens.

'n Aangewysde praktisyn wat nie persoonlik deur die uitslag van die outopsie-ondersoek geraak word nie, is veel beter in staat om die ondersoek uit 'n onpartydige oogpunt te betrag. Daarenteen kan 'n beskuldigde (of 'n praktisyn wat later 'n beskuldigde kan word) 'n baie wesenlike belang daarby hê om wat in die loop van die ondersoek gesien en gedoen is by 'n latere verhoor te betwis of miskien verkeerd voor te stel.

'n Uiterste voorbeeld van wat in sulke omstandighede kan gebeur, word verstrek deur die berugte saak van dr. Palmer, 'n mediese praktisyn wat ‚sy professie versaak en 'n wedder op perdewedrenne geword het, in die loop waarvan hy intieme betrekkinge aangeknoop het

necessary permission. As there is no greater authority vested in one or other of these officers, there is the theoretical possibility of a conflict between the magistrate and the medical practitioner.

Apart from the permission which may be granted by the magistrate or the medical practitioner, each exercising his own discretion, there is a right granted to certain persons to be present which does not depend on the discretion of the magistrate or the medical practitioner concerned. These 2 classes to the exception of the general provision are:

(*a*) Policemen.

(*b*) Medical practitioners nominated by persons who have a substantial and peculiar interest in the issue of the examination, provided they have satisfied the magistrate that they have that interest.

A certain obscurity is created by the use of the term 'nominate.' It is not clear whether, if the person with a substantial and peculiar interest is a medical practitioner, such a practitioner can nominate himself. The provision makes it definite that citizens generally cannot be present in person. It would seem, therefore, that the fact that the citizen concerned happens to be a medical practitioner would not give him rights greater than those of other citizens. On this construction such a practitioner would have to nominate some other practitioner to represent him. Apart from the inherent propriety of this procedure, it is desirable for other reasons.

Where the inquiry is into a cause of death where there was unlawful conduct, the medical practitioner may have been the person responsible, e.g. in the case of death associated with anaesthesia, an abortion, murder, etc.

A nominated practitioner who is not personally concerned with the result of the autopsy examination is in a much better position to view the examination dispassionately; whereas an accused person (or a practitioner who may become an accused person) would have a very real interest in disputing and perhaps misrepresenting at the subsequent trial what was, in fact, seen and done.

The extreme example of what can occur in such circumstances arose in the notorious case of Dr. Palmer, a medical practitioner who had 'given up his profession for the pursuits of the Turf, in the course of which he became intimate with a young man named Cook, who was addicted to the same pursuits.'[1] Dr. Palmer, with the object of substantial gain from Cook's death in order to retrieve his own catastrophic position, murdered Cook by poisoning with strychnine. Dr. Palmer had also prevailed upon the medical practitioner

met 'n jong man met die naam van Cook wat ook in weddenskappe belang gestel het.'[1] Met die doel om aansienlike geldelike voordeel uit Cook se dood te trek, en hom daardeur uit sy eie benarde posisie te verlos, het dr. Palmer Cook vermoor deur hom met strignien te vergiftig. Dr. Palmer het die mediese praktisyn wat die oorledene behandel en reeds 'n gevorderde leeftyd bereik het, oorreed om te sertifiseer dat Cook aan apopleksie oorlede is.

,'n Geregtelike lykskouing is onderneem wat deur die beskuldigde (dr. Palmer) bygewoon is. Die maag en ingewande is in 'n fles geplaas met die doel om dit vir ondersoek na Londen te stuur. Onderwyl die operasie nog aan die gang was, het die gevangene teen die mediese manne wat daarby betrokke was, gestamp met die gevolg dat 'n gedeelte van die inhoud van die maag in die lyk te lande gekom het. Die fles is toe met perkament bedek, vasgemaak en verseël, en een kant neergesit. Onderwyl die aandag van die mediese manne nog op die ondersoek van die lyk toegespits was, het die gevangene (dr. Palmer) die fles verskuif, tot naby 'n deur wat nie die gewone uitgang was nie, en daar is later bevind dat twee gleufies gesny is in die dubbele vel waaruit die bedekking van die fles bestaan het. Die gevangene wat verneem het dat die fles daardie selfde aand na Londen gestuur sou word, het die koetsier wat die persone in bevel van die fles na die stasie sou neem, £10 aangebied om die koets te laat omslaan en die fles te breek. Die ontleders na wie die maag en ingewande en later ook ander dele van die lyk gestuur is, het spore van antimoon gevind, maar geen strignien of ander gif nie, en het hul verslag per pos aan die prokureur wat by die ondersoek betrokke was op Rugeley gestuur. Die gevangene het die posmeester oorgehaal om die inhoud van hierdie verslag aan hom te openbaar en het toe 'n vertroulike brief aan die lykskouer geskryf (aan wie hy in die loop van die ondersoek geskenke van vis en wildsvleis gestuur het) om te sê dat hy in swart en wit gesien het dat geen strignien, blousuur of opium gevind is nie. Hy het ook die hoop uitgespreek dat op die volgende dag, wanneer die geregtelike lykskouing hervat sou word, die uitspraak ‚dood ten gevolge van natuurlike oorsake' sou wees. Die lykskouingsjurie se uitspraak was dat opsetlike moord deur die gevangene gespleeg is.'[2]

PERSONE WAT DIE REG HET OM BY 'N LYKSKOUING VERTEENWOORDIG TE WORD

Hierdie persone word duidelik omskryf as persone wat 'n wesenlike en besondere belang by die uitslag van die ondersoek het. Die saak van die Prokureur-generaal *vs.* Van der Merwe en Bornman, 1946, O.P.A., 197 op bladsy 201, suggereer dat die behoorlike vertolking van hierdie frase is dat die betrokke persoon in 'n ander mate deur die uitslag van die ondersoek geraak moet word as enige ander lid van die publiek. Op hierdie grondslag sal blote opvoedkundige of wetenskaplike belang in die uit-

1. *Wills' Principles of Circumstantial Evidence*, 7e uitgawe, Londen: Butterworth & Co. (Publishers) Ltd., 1936, bl. 406.
2. *Op cit.*, bladsye 410-411.
Trial of William Palmer for Murder by Poisoning. (Notable British Trials Series). 3de uitg., 1952, bl. 42. Gered. deur George H. Knott. Nagesien deur Eric R. Watson. Londen en Edinburgh: William Hodge and Company, Limited.

who had attended the deceased, and who was of a very advanced age, to certify that he had died of apoplexy.

'A post-mortem examination was made, at which the prisoner (Dr. Palmer) was present, and the stomach and intestines were placed in a jar to be taken to London for examination. While the operation was going on, the prisoner pushed against the medical men engaged in it, so as to shake a portion of the contents of the stomach into the body. The jar was then covered with parchment, tied down, and sealed and placed aside; and while the attention of the medical men was still engaged in examining the body, the prisoner (Dr. Palmer) removed the jar to a distance, near a door not the usual way out of the room, and it was found that two slits had been cut through the double skin forming the covering. The prisoner having learned that the jar was to be sent to London the same evening, offered the driver who was to carry the persons in charge of it to the railway station £10 to upset the carriage and break the jar. The analysts, to whom the stomach and intestines, and, subsequently, other parts of the body, were sent, found traces of antimony, but none of strychnia, or any other poison; and sent their report by post, directed to the attorney at Rugeley employed in the investigation. The prisoner incited the postmaster to betray to him the contents of this report; and wrote a confidential letter to the coroner, to whom during the course of the inquiry he sent presents of fish and game, stating that he had seen it in black and white that no strychnia, prussic acid, or opium had been found, and expressing his hope that on the next day to which the inquest stood adjourned, the verdict would be that of death from natural causes. The coroner's jury found a verdict of wilful murder against the prisoner.[2]

PERSONS WHO HAVE A RIGHT TO BE REPRESENTED AT THE AUTOPSY

These persons are clearly defined as those who have a substantial and peculiar interest in the outcome of the examination. The case of Attorney-General *vs.* van der Merwe & Bornman, 1946, O.P.D., 197 at p. 201, suggests that a proper interpretation of this phrase is that the person concerned must be affected by the issue of the examination in a different degree from the way in which any other member of the public may be affected. On this construction mere educational or scientific interest in the outcome of a post-mortem examination would not constitute a substantial and peculiar interest. The educational and scientific needs of medical practitioners and medical students are satisfied through the exer-

1. *Wills' Principles of Circumstantial Evidence*, 7th ed., London: Butterworth & Co. (Publishers) Ltd., 1936, p. 406.
2. *Op cit.*, pp. 410-11.
Trial of William Palmer for Murder by Poisoning. (Notable British Trials Series). 3rd ed., 1952, p. 42. Ed. by George H. Knott. Revised by Eric R. Watson. London and Edinburgh: William Hodge and Company Limited.

slag van 'n geregtelike lykskouing nie op wesenlike en besondere belang neerkom nie. In die opvoedkundige en wetenskaplike behoeftes van mediese praktisyns en mediese studente kan daar voorsien word deur die uitoefening van diskresie deur die mediese praktisyn wat die outopsie waarneem.

Afgesien van persone wat by kriminele sake betrokke kan wees, is daar ook heel duidelik 'n ander klas persone* met belang in moontlike siviele gedingvoering wat 'n wesenlike en besondere belang by die uitslag van die ondersoek kan hê. Gevolglik is dit raadsaam vir die mediese praktisyn om nie sy algemene diskresie te gebruik as hy 'n versoek ontvang van 'n persoon wat 'n magistraat klaarblyklik tevrede moet stel vir sover dit sy wesenlike en besondere belang betref nie.

In die praktyk kan moeilikhede geopper word deur die polisie op wie se persele die outopsie gewoonlik plaasvind. Hulle kan aandring op 'n lasbrief van die magistraat wat die benoemde of ander praktisyns magtig om teenwoordig te wees. As verlof egter eenmaal deur die magistraat of die betrokke mediese praktisyn toegestaan is, het 'n persoon 'n statutêre reg om teenwoordig te wees, en hierdie reg weeg swaarder as enige departementele polisieopdrag in die onderhawige saak.

* in die oë van die wet sluit ‚persone' waarskynlik 'n boedel, 'n korporatiewe liggaam, ens., in.

cise of his discretion by the medical practitioner conducting the autopsy.

Apart from persons concerned with criminal matters, there is clearly another class of persons* with an interest in possible civil litigation, who may have a substantial and peculiar interest in the issue of the examination. Hence a medical practitioner would be well advised to exclude from the exercise of his general discretion any request from any person who clearly needs to satisfy a magistrate about his substantial and peculiar interest.

In practice difficulties may be raised by the police, on whose property the autopsy is usually carried out. They may require an order from the magistrate for the nominated or other practitioners to be present. Once, however, permission has been given by the magistrate or the medical practitioner concerned, that person has a statutory right to be present, overruling any departmental police directive in the matter.

* 'Person' probably includes in law an estate, a body corporate, etc.

THE CORONARY ARTERIAL PATTERN IN BANTU AND EUROPEAN HEARTS*

FURTHER ANATOMICAL VARIATIONS

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In his original study of European, Bantu and Coloured hearts, Brink[1] suggested that the coronary arterial pattern of the Bantu heart differs significantly from that of the European, in that the former frequently shows a third primary division of the left coronary artery. Although some doubt was cast on this finding by Singer,[2] Pepler and Meyer[3] subsequently demonstrated this vessel to be present in 74% of Bantu as compared to 38% of European hearts. Brink,[1] however, also drew attention to other peculiarities of the Bantu circulation, viz. a high termination of the anterior descending branch of the left coronary artery and so-called 'right coronary preponderance.'

The pathological and clinical significance of the 'right coronary preponderant' heart has been pointed out by Schlesinger.[4] The importance of a high terminating descending branch of the left coronary artery is difficult to assess at present. In view of the better anastomotic circulation in Bantu hearts,[3, 5] it is necessary to determine whether the presence or absence of such a vessel bears any relation to the better circulation.

The purpose of the present paper is to compare Bantu and European hearts with regard to the presence or absence of a high terminating left descending branch and at the same time to try to correlate the presence of such an anatomical variation with the better anastomotic supply of the Bantu heart.

MATERIAL

The material used was essentially the same as that described previously,[3] although 4 Bantu hearts were left out in the present series because of inadequate injection of the right coronary artery tree.

The age and sex distribution of the series is given in Table 1.

METHODS

The X-ray films were labelled in such a way that it was not possible to identify the racial group of the case and each film was read separately by both authors.

* The expenses for this work have been defrayed by a grant from the Council for Scientific and Industrial Research.

A long descending or so-called high terminating, descending branch of the left coronary artery was regarded as present when this branch extended for a third, or more than a third, of the way up the posterior interventricular groove.

The criteria for labelling a heart as belonging to the 'right preponderant,' 'left preponderant' or 'balanced circulation' type were as described by Schlesinger.[4]

Note was also made of the termination of the posterior interventricular branch of the right coronary artery in all those cases where this branch extended right to the apex. At the same time, attention was given to major variations of the origin or primary branches of the right coronary artery, such as a double artery or immediate division into two major branches.

TABLE 1: AGE AND SEX DISTRIBUTION OF THE SERIES

Age Group (years)	*European*		*Bantu*	
	Male	*Female*	*Male*	*Female*
0— 9	2	4	2	8
10—19	7	3	4	8
20—29	7	3	6	4
30—39	5	6	6	5
40—49	5	5	9	5
50—59	5	5	5	5
60—69	4	6	6	5
70—79	8	4	5	3
80—89	7	4	4	4
Total	50	40	47	47

RESULTS

Type of Circulation. The distribution of the types of circulation according to Schlesinger[4] is given in Table 2. Illustrations of 'left coronary preponderant' and 'right coronary preponderant' circulations are shown in Figs. 1 and 2.

TABLE 2: DISTRIBUTION OF TYPE OF CIRCULATION IN BANTU AND EUROPEAN HEARTS

	European	*Bantu*	*Total*
Balanced circulation	28	43	71
Left preponderance	17	8	25
Right preponderance	45	43	88

TABLE 3: INCIDENCE OF A HIGH TERMINATING LEFT DESCENDING CORONARY ARTERY IN THE VARIOUS TYPES OF CIRCULATION

	High Terminating Left Descending Coronary Artery	*European*	*Bantu*
Total	56	15	41
Balanced	33	9	24
Right Preponderant	12	2	10
Left Preponderant	11	4	7

High Terminating Left Descending Coronary Artery. The frequency of this artery in the series and its relation to the various types of circulation is given in Table 3. A typical example is shown in Fig. 3. The association of a high terminating left descending branch and its association with intercoronary anastomoses is given in Table 4.

TABLE 4: HIGH TERMINATING LEFT DESCENDING CORONARY ARTERY AND INTERCORONARY ANASTOMOSES

	Total	*European*	*Bantu*
With communication	37	8	29
Without communication	19	9	10

DISCUSSION

The most significant finding in the present series is the presence of a high terminating left descending coronary artery in 56 of 184 cases (30%). Of these, 41 were found in Bantu hearts (44%) and only 15 in European hearts (17%). It seems, therefore, that this type of artery occurs much more frequently in the Bantu than in the European heart, and is thus in accordance with Brink's[1] original finding. Analysis of Table 4, makes it apparent that the presence of this artery is associated with higher occurrence of intercoronary anastomoses in Bantu than in European hearts. Whether this single vessel plays any important role in the causation of a better anastomotic circulation, will need further proof.

An attempt was also made to associate the presence of a long descending or high terminating left descending coronary artery with the third primary division of the left coronary artery. Of a total of 56 cases, only 30 cases (65%) showed a third primary division of the left coronary artery. It is therefore obvious that this does not occur with any greater frequency than in the general series.

Analysis of the types of circulation (Table 2), makes it apparent that the Bantu has a much higher frequency of a so-called 'balanced circulation' than has the European, and that the European a much more frequently occurring 'left preponderant' circulation. It has been pointed out by Schlesinger[4] that these 'balanced circulation' hearts suffer less from the effects of coronary atherosclerosis than do the other 2 types.

Brink[1] has suggested that the Bantu heart shows a higher frequency of a 'right preponderant' circulation, but this could not be confirmed in the present series as, of a total of 88 cases with this type of circulation, 43 occurred in Bantu hearts and 45 in European hearts.

An analysis of the cases where the right posterior interventricular artery extends as far as the apex of the heart shows, as could have been expected, an association of this vessel with a 'right preponderant' circulation, but no difference could be demonstrated between the 2 racial groups.

2. It is shown that the Bantu possesses a much more frequently occurring high terminating left descending coronary artery.

3. The Bantu heart shows a preponderance of the so-called 'balanced type' of circulation.

We are indebted to Mr. C. J. de Swardt for the photomacrographs.

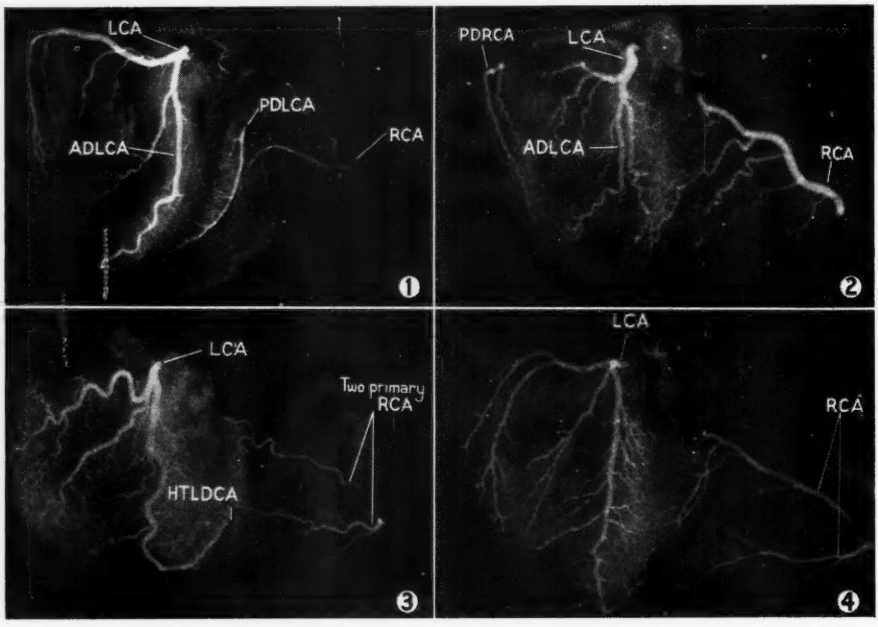

Fig. 1. 'Left coronary preponderant' circulation in a European male aged 18 years.
Fig. 2. 'Right coronary preponderant' circulation in a European female aged 35 years.
Fig. 3. High terminating left descending coronary artery and 2 primary right coronary arteries in a Bantu male aged 45 years.
Fig. 4. Immediate division of the right coronary artery in a European female aged 18 years.

LCA:	Left coronary artery	RCA:	Right coronary artery
ADLCA:	Anterior descending branch of the left coronary artery	PDLCA:	Posterior descending branch of the left coronary artery
PDRCA:	Posterior descending branch of the right coronary artery	HTLDCA:	High terminating left descending coronary artery

A further interesting finding was the frequency of an abnormal origin or early division of the right coronary artery into 2 major branches (Fig. 4). This occurred in 11 of the 184 cases examined, of which 6 were found in Bantu hearts and 5 in European hearts. A single left coronary artery was present in one case.

SUMMARY

1. A series of 184 Bantu and European hearts was analysed with regard to certain anatomical variations of the coronary artery pattern.

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THE TREATMENT OF HYPERTONIC DEHYDRATION

PART 2

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Balance studies during the development as well as during the correction of hypertonic dehydration[1, 2] have shown that water depletion is the predominant disturbance of the body fluids. Two thirds of the water loss is derived from the intracellular compartment and one third from the extracellular compartment (Fig. 1). In

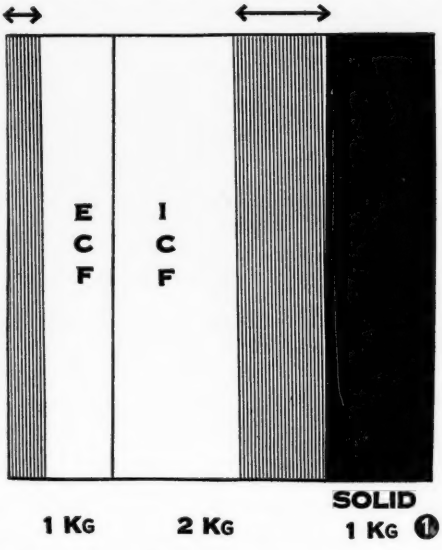

Fig. 1. Body Fluid Compartments in a 4-Kg. child with hypertonic dehydration.
The shaded areas represent the water lost from the intracellular fluid (ICF, $-\frac{2}{3}$) and the extracellular fluid (ECF, $-\frac{1}{3}$).

experimental hypernatraemic dehydration tissues in various sites reacted differently to the surrounding hypertonic extracellular fluid. To maintain osmotic equilibrium in muscle, water moved out while there was a shift of sodium into the cells. In contrast, sodium did not enter the cells of the brain. Instead, there was a considerably greater dissociation of intracellular complex particles.[3] This ionic disturbance in the brain cells probably accounts for most of the nervous manifestations observed in children with hypertonic dehydration. In a few instances these disturbances may be due to intracranial haemorrhage and/or subdural effusions, both of which have been seen in clinical and experimental hypertonic dehydration.[3, 4]

There is virtually no deficit of chloride in hypertonic dehydration. On the other hand, there has usually been a loss of potassium from the body via the diarrhoeal stools. Hypocalcaemia is found not infrequently.[1] This was detected in 3 of the 39 patients previously reported by us.[5] The serum calcium levels were 3.1, 3.4, and 3.7 mEq. per litre. Two of these infants had tetany while the third child developed convulsive twitchings.

From these considerations of the physiology and pathology of hypertonic dehydration, it would appear logical to treat this condition by replacement with water alone. However, experience has shown that massive and rapid infusion with water precipitates another disturbance of the osmotic equilibrium and convulsions frequently result. Furthermore, cardiac failure may supervene from too rapid expansion of the extracellular compartment. The raised serum sodium level in hypertonic dehydration reflects a diminution in plasma water and not an absolute increase in serum sodium. In fact, there is a reduction in the absolute amount of sodium in the extracellular fluid due to some loss of this element in the stools. Addition of a small amount of sodium is therefore necessary in the rehydrating solution, to allow for a more physiological restoration of the osmotic equilibrium between intracellular and extracellular compartments. Convulsions are then less likely to occur. It is recommended that an intravenous infusion of 40 ml. molar sodium lactate dissolved in 1,000 ml. of 5% glucose in water (sodium: 40 mEq. per litre) be administered. This gives a final concentration of M/25 sodium lactate. The total volume

* Paper read at the *Symposium on Parenteral Fluid Therapy* held in Johannesburg on 29 October 1960.

of fluid given in 24 hours is 80–100 ml. per lb. body weight. If molar sodium lactate is not available, a solution containing 0.2% sodium chloride in 5% invert sugar (sodium: 34 mEq. per litre) may be used.

The metabolic acidosis which is commonly found requires no specific therapy. An alkaline solution such as M/6 sodium lactate should be avoided because of its high sodium content, viz. 167 mEq. per litre. With restoration of the fluid deficit, renal function returns and, with it, correction of the acidosis. Similarly, the high rectal temperatures which often occur in this condition settle within 24–36 hours after commencement of intravenous therapy.

As oliguria or even anuria is a common presenting feature in hypertonic dehydration, potassium replacement is carried out only when diuresis commences. Potassium chloride or potassium acetate (26 mEq.) is then added to each 1,000 ml. of fluid.

Daily electrolyte estimations are performed and milk feeds are not given until the serum electrolyte levels return to normal. The reasons for this are twofold. Firstly, a high intake of food tends to increase the bulk of stools and hence increases the water loss via this route.[6] The second, and more important, reason is that the relatively high protein and electrolyte content of milk increases the solute load on the kidney.[7] This results in a greater obligatory water loss via the kidneys. For example, for every gramme of protein not utilized by the body, 20 ml. of extra water are required by the kidneys for the excretion of the products of catabolism from this protein.[8] Thus an infant already dehydrated from gastro-enteritis and unable to utilize protein feeds, has a smaller margin of safety for water conservation. When milk feeds are re-introduced, it is suggested that a formula consisting of 2 parts milk and 1 part water be used. Skim milk formulae should be avoided as there is a greater load of protein and electrolyte per calorie of feed than with two thirds milk.

Hypocalcaemia, when it occurs, is treated by the addition of 10–30 ml. of a 10% calcium gluconate solution to the infusion.

If the nervous system manifestations persist, and hypocalcaemia has been excluded, the presence of subdural effusions should be suspected.[4] Subdural taps are then carried out.

There have been reports of infants suffering from gastro-enteritis who developed hypertonic dehydration because they were inadvertently given concentrated electrolyte solutions by mouth.[1, 7] If an oral solution is to be administered, then it should be prepared in the correct dilution. At this hospital there is a stock concentrated electrolyte solution containing sodium chloride 4 grains (0.25 g.) and potassium citrate 6 grains (0.4 g.) per drachm. The mother is instructed to add one teaspoon of this solution to 4 oz. (120 ml.) boiled water. This gives a 0.25 N sodium chloride solution.

The underlying cause of the hypertonic dehydration will require treatment. The commonest cause in our series of infants was summer diarrhoea. This was treated with chloramphenicol (chloromycetin palmitate) 40–50 mg. per lb. body weight per 24 hours and was given in divided doses every 6–8 hours.[9] Respiratory infection occurred primarily or was associated with the diarrhoea. Penicillin was given to these patients by intramuscular injection in doses of 250,000 units every 6 hours. We have seen a few cases of meningitis with hypertonic dehydration. The antibiotics used depended upon the organism isolated from the cerebrospinal fluid.

With this therapeutic regimen the mortality rate in the well nourished group of infants in our series was 9.7%. This compares favourably with the mortality rate of 11.6% among a group of American children who were treated for hypertonic dehydration.[1]

SUMMARY

The rationale and details of treatment of hypertonic dehydration and its complications are considered.

We thank Dr. I. Frack, Superintendent, Baragwanath Hospital, for permission to submit this paper for publication.

We are indebted to Drs. E. Kahn and S. Wayburne for facilities to study cases under their care; the South African Institute for Medical Research for the electrolyte estimations; and the Photographic Unit, Department of Medicine, University of the Witwatersrand for Fig. 1.

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PRIMARY MURAL ENDOCARDIAL DISEASE

A DISCUSSION OF THE CONDITION AND A REPORT OF A CASE IN A WHITE MALE AGED 54 YEARS

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Primary idiopathic mural endocardial disease is a condition that has amassed a voluminous literature and a multiplicity of names. The description of the first case is ascribed to Kreysig[1] in 1818, and Fisher[2] in 1911, was one of the first to stress the salient histological features.

Rosahn[3] states that since 1950 the name endocardial fibro-elastosis appears to be a generally accepted term for primary mural endocardial disease. Other frequently used synonyms include congenital endocardial fibro-elastosis,[4] adult endocardial fibro-elastosis,[5] fibroplastic myocarditis,[6] constrictive endocarditis,[7] parietal endocarditis,[8] fibroplastic parietal endocarditis,[9] endomyocardial fibrosis,[10, 11] endomyocardial sclerosis,[12] cardio-vascular collagenosis with parietal thrombi,[13] the malnutrition heart,[14] cryptogenic heart disease,[15] some forms of the pregnancy heart,[16, 17] fibrosis of the endocardium and myocardium with mural thrombosis,[18] endocarditis obliterans.[19] In addition, Gowing[20] has stressed that primary mural endocardial disease is the important factor in the so-called idiopathic cardiac hypertrophy, and Kugel and Stoloff[21] claim that congenital idiopathic hypertrophy of the heart has now been shown to be a very rare disease. In their review of 852 cases, 17 only were regarded by those authors as probable examples of congenital idiopathic hypertrophy. Of these cases, many had endocardial sclerosis and bland thrombi on the ventricular endocardium. Mannheimer *et al.*[22] described a case of pure mitral stenosis in an infant due to primary mural endocardial disease and seriously question whether isolated congenital mitral stenosis is due to a disturbance of development.

All the conditions listed are examples of primary damage to the mural endocardium of the heart of unknown etiology and should, in our present state of ignorance, be grouped together and referred to as primary mural endocardial disease. Davies[23] (in his recent, excellent review of some of the idiopathic lesions of the mural endocardium) states that 'world medicine is made up of lumpers and splitters' and he obviously belongs to the latter group. At this stage, when the etiology of the condition is unknown, it is more reasonable to be a 'grouper' and to subdivide later, on an etiological basis, when various causes of this condition become known.

It must of course be clearly understood that all secondary causes of mural endocardial disease must be eliminated before a diagnosis of primary endocardial disease is made. Such causes of secondary mural endocardial disease include congenital anomalies of the cardiovascular system, myocarditis of various types, e.g. viral, rickettsial, bacterial, parasitic, chemical and idiopathic, rheumatic and rheumatoid disease, generalized lupus erythematosis, periarteritis nodosa, the carcinoid syndrome, scleroderma, healed infarctions, generalized glycogenesis[24] and gargoylism.[25]

The lesion in the mural endocardium in the secondary type is usually mild. In some parts of the world, e.g. Africa, primary mural endocardial disease is the commonest form of idiopathic heart disease. As has also been stressed by Panke and Rottimo,[26] it can easily be overlooked by the uninformed pathologist, particularly when it presents in the acute and subacute stages.

Many cases have been inadequately investigated from the pathologic aspect. Blumberg and Lyon[27] state that in their experience the condition is the commonest cause of fatal heart disease in infancy, and Lambert *et al.*[28] found 15 cases in 2,352 consecutive autopsies on infants and children. Bali *et al.*[11] consider it a major cause of heart disease in Uganda and elsewhere in Africa. Davies[10] states that 10% of African cases dying of congestive heart failure in Kampala were examples of this condition and found an autopsy incidence of 0.95%. Becker *et al.*[13] report on autopsy incidence in Johannesburg, South Africa, of 0.71% in blacks and 0.18% in whites. Elsewhere

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lower incidences have been reported, e.g. Panke and Rottimo[26] found one case in 3,500 adult autopsies. As Blumberg and Lyon[27] have stated, quite correctly, the perplexing terminology has led to confusion, and until this is satisfactorily settled one can only agree with Rosahn[3] when he states that the true frequency of this disease may never be satisfactorily determined.

ETIOLOGY

All ages are affected.[11, 13, 29, 30] Both black and white races are affected,[13] but it appears to be commoner in the former.[11, 13] Many etiological mechanisms have been suggested, without conclusive results, e.g. genetic and developmental origin,[3, 20, 31] infective,[32] virus,[33] malnutrition,[14] enzyme deficiency,[4] auto-immune disease,[13, 34] allergic,[8, 9] anoxia,[35] endocrine disturbance.[16] In a recent publication Higginson *et al.*[15] postulate a basic myocardial weakness of unknown etiology. Obviously much more research will be needed before etiological consideration can be placed on a definitive basis.

THE PATHOLOGY AND PATHOGENESIS OF PRIMARY MURAL ENDOCARDIAL DISEASE

The disease may present itself in the acute, subacute or chronic stage and all stages may be present in one individual. It is characteristically a recurrent and focal disease. Dennis *et al.*[31] reviewed the autopsy findings of 149 infants and children and showed that 25% were of the fulminating type and were under 6 weeks of age, 51% were acute cases, ages 6 weeks to 6 months, and 21% were chronic cases in children and older infants; 32.5% of the cases of Becker *et al.*[13] were of the acute variety, 62.5% were in the subacute stage of the disease and only 5% were early chronic cases. The severe diffuse chronic type was not seen in their series.

In the vast majority of cases the heart is heavier than normal and hypertrophy and dilatation of all chambers is usually present. It must, however, be emphasized that hypertrophy is not an essential feature.[13] Some stress that the right side is more affected, but as separate segmental weighings of the ventricles have not been reported in this condition, this cannot be accepted. The normal mural endocardium has virtually no thickness macroscopically and is translucent except in the auricles and below the aortic valves, where it appears opaque. Cosgrave and Kaump[36] state correctly that the gross pathological findings are variable and depend on the stage of the disease.

The mildest forms of the chronic stage show focal opacities varying in size and situated anywhere in the mural endocardium. Mixed antemortem thrombi may or may not be attached to the thickened endocardium. It must be stressed that, although the lesion may not be extensive, severe functional changes may occur. Histologically the thickened patches show a fibroelastosis and the subendocardial muscle in the affected areas shows varying degrees of ischaemic change. The rest of the myocardium is normal.

The severest forms of the chronic stage show gross diffuse thickening of the mural endocardium, commonly affecting the left ventricle. The papillary muscles and chordae tendineae are embedded in the collagen mass. Mixed ante-mortem thrombi may or may not be present on the fibrosed endocardium and may be found in the appendices of the auricles. Gray[33] states that if mural thrombi do not occur the disease may last for years. Adhesions may occur between the mitral and tricuspid valves and the underlying endocardium and fibrosis and distortion of one or more valves may or may not be found. Smooth or warty nodules may occur on any surface of any valve. The thickened endocardium may have a rugose appearance and the fibrosis may be so marked at the apex of the heart as to cause retraction in this area.[11] The underlying myocardium shows a varying degree of ischaemic fibrosis. Bedford and Konstam[37] liken this subendocardial fibrosis to shallow infarcts. The coronary arteries appear normal and the pericardium is unaffected.

Microscopically, the striking feature is a gross fibrosis of the mural endocardium, with varying degrees of elastosis. Fibrin deposits are frequently noted on the fibrosed endocardium. Still and Boult[38] studied the fibrin deposits by electron microscopy and state that the incorporation of the fibrin deposits is an important factor in the endocardial fibrosis. These deposits become altered to a collagen-like substance and may be seen embedded deep in the collagen mass.[29, 32] Organization of the mixed antemortem thrombi occurs, increasing the fibrosis of the endocardium. The valvular lesions are similar to those in the mural endocardium. The subendocardial muscle shows an ischaemic fibrosis due to narrowing and obliteration of the mouths of the thebesian vessels.[26, 40] A non-specific, slight, non-granular celled infiltration may be found and in addition Davies[10] and Löffler[9] have described eosinophils. Gray[33] considered that the myocardial fibrosis was due to embolization of the coronary vessels by platelets and fibrin and noted that the intramural thebesian vessels contained recanalized thrombi. The majority of cases

reported show the severe chronic stage of the disease.

In the acute and subacute stages the endocardium shows slight focal thickenings anywhere in the mural endocardium and antemortem thrombi are frequently found on the damaged areas. Becker *et al.*[13] state that this is the characteristic finding and the commonest site of the lesion is between the papillary muscles at the apex of the left ventricle and varies in degree. Subendocardial necrosis and fibrosis of the myocardium occur beneath the thickened endocardium. Histologically the earliest change is a focal swelling of the endocardium due to mucoid oedema which is metachromic when stained with toluidene blue.[3, 13] Deposits of fibrinoid material may be found in addition.[13, 34] This mucoid oedema is a characteristic finding and the commonest site of the lesion is between the papillary muscles of the left ventricular apex.[13] Mixed ante-mortem thrombi are frequently found on the damaged endocardium. Subendocardial myocardial degeneration occurs and varies from hydropic degeneration and enlargement and hyperchromatism of the muscle nuclei to necrosis and later fibrosis. A non-specific, non-granular celled infiltrate may be found in these areas. Later collagenosis and elastosis occur. Valvular lesions are not usually noticed in the acute, subacute and early chronic stages.

In addition to the cardiac lesions, vascular lesions have been described in the aorta and the visceral vessels.[3, 13, 41, 42]

At no stage of the disease can the lesions be interpreted as inflammatory.

A REPORT OF A CASE OF PRIMARY MURAL ENDOCARDIAL DISEASE

1. CLINICAL FINDINGS

A 53-year-old white ex-miner was admitted to the Johannesburg General Hospital on 10 March 1959, complaining of increasing breathlessness for 2 years. This had been rapidly progressive. He had had to sleep on at least 5 pillows for the past year and swelling of the ankles had developed 4 months before admission. There was an associated unproductive cough but he had had no chest pain. The patient stated that he had been completely well until October 1944, when he developed a left pleural effusion while on active service in Italy. This had been diagnosed as tuberculosis and he had been sent back to this country where he remained in a sanatorium throughout 1945. *M. tuberculosis* was found in the sputum on several occasions until August 1945, but never since. A left phrenic crush was performed in September 1945. He was discharged early in 1946 and had remained well until the onset of the present illness.

He was first seen by one of us (J. B. B.) on 24 March 1959. Examination at that time revealed that he was dyspnoeic at rest with a respiration rate of 40 per minute. He was also extremely orthopnoeic. There was slight oedema of the ankles and sacrum but no clubbing, anaemia or jaundice. The extremities showed some cyanosis but the tongue appeared pink. A left Horner's syndrome, which had evidently developed after the phrenic crush in 1945, was present. The pulse rate was 90 per minute, regular and of normal volume. The blood pressure was 130/70 mm. Hg and a paradoxical pulse was noted from 130 down to 110 mm. Hg. The jugular venous pressure was raised 15 cm. and a prominent systolic wave of tricuspid incompetence was clearly seen. The apex beat could just be felt in the 5th intercostal space in the mid-clavicular line. There was a marked right ventricular heave at the sternum, but no clinical evidence of left ventricular hypertrophy.

On auscultation a grade 2 pansystolic murmur[43] was heard on deep inspiration at the lower sternal border, which was consistent with the regurgitant systolic murmur of tricuspid incompetence. A summation 'gallop' was also audible during inspiration at this site and thus presumably arose from the right ventricle.[44] The second heart sound was narrowly split, the pulmonary component was increased in intensity and its timing in relation to the aortic component was unaffected by respiration. The left hemithorax moved less than the right and there was impairment of percussion and decrease in the breath sounds on that side. No adventitious sounds were heard in the lungs.

On palpation of the abdomen, the liver was enlarged 5 finger-breadths below the right costal margin and direct pulsation of this organ was detected.

An electrocardiogram showed marked change from one which had been done during a routine examination in 1956 which was considered normal (Fig. 1). Clockwise rotation had now developed and a QS pattern over the right precordial leads was compatible with right ventricular dilatation. Left atrial enlargement was suggested by the wide negative P wave in Lead 6.[45]

A postero-anterior radiograph (Fig. 2) showed some cardiomegaly (cardio-thoracic ratio 53%). There was decreased translucency

of the left lung field due to pleural thickening. An oval opacity in the left upper zone had the appearances of an old fibrotic tuberculous lesion. A diffuse granular mottling in both lower zones and prominent superior pulmonary veins suggested a raised pulmonary venous pressure. Fullness in the region of the left

A blood count on 13 March 1959, showed haemoglobin 17.3 g. %; white cell count, 5,000 per c.mm. (Neutrophils: 77%; Monocytes: 7%; Lymphocytes: 14%; Eosinophils: 1%; Basophils: 1%). The erythrocyte sedimentation rate was 4 mm. in the first hour (Wintrobe).

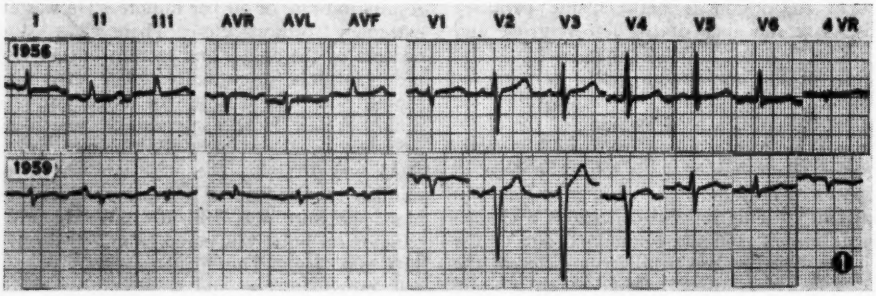

Fig. 1. The electrocardiograms taken in 1956 and 1959. The 1959 tracing shows clockwise rotation, left atrial enlargement and a probable right ventricular dilatation.

atrial appendage was compatible with left atrial dilatation and this was confirmed by a barium swallow in the right oblique position (Fig. 3). On fluoroscopy (Dr. H. Jackson) pulsation of the heart borders was of small amplitude but was present throughout.

The features were thus those of pulmonary hypertension with right ventricular failure and tricuspid incompetence. There seemed insufficent clinical evidence of pulmonary disease to cause this degree of right ventricular failure and this was largely confirmed by lung function

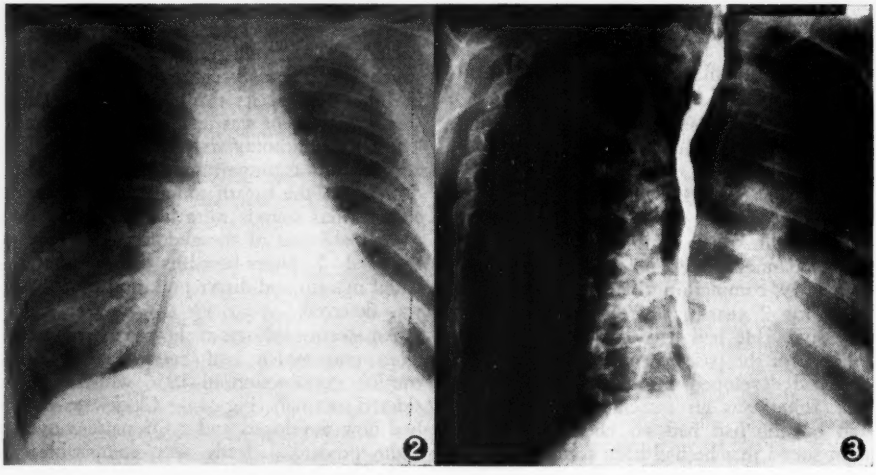

Fig. 2. A postero-anterior radiograph showing cardiomegaly, decreased translucency of the left lung field due to pleural thickening, an oval opacity at the left apex, a prominent left atrial appendage and pulmonary 'congestion'.

Fig. 3. A barium swallow in the right oblique position showing backward displacement of the oesophagus due to left atrial enlargement.

tests (Dr. S. Zwi) which, while showing a degree of obstructive and restrictive lung disease, were thought to show an important cardiac factor to account for the undoubted severely impaired exercise tolerance. The extreme orthopnoea and the radiological signs of pulmonary venous hypertension with probable left atrial enlargement suggested a left-sided cause for the right ventricular failure. The absence of an apical mid-diastolic murmur, evidence of congestive cardiac failure with sinus rhythm and electrocardiographic signs of marked right ventricular hypertrophy,[46] all virtually excluded mitral stenosis; but a left atrial myxoma or chronic left ventricular failure were possible diagnoses. Partial obstruction of the pulmonary veins[47-50] following tuberculous involvement of the mediastinum, was also considered. Significant constrictive pericarditis was considered unlikely.

Fig. 4. A postero-anterior angio-cardiogram with a catheter placed in the main pulmonary artery. The film was taken 14 seconds after the injection of the dye. The distended pulmonary veins and opacified left atrium and left ventricle can be seen. The left ventricular wall appears to be unduly thick.

The patient improved slightly on digitalis and diuretic therapy and a cardiac catheterization was done on 9 June 1959. The pulmonary artery pressure was 95/45 mm. Hg and the right ventricular pressure 95/5 mm. Hg. The typical dip and plateau pattern of constriction[51] was not seen on the right ventricular pressure tracing. A pulmonary wedge pressure was unfortunately not obtained.

An angiogram was performed from the main pulmonary artery, which showed large pulmonary arterial branches, though there was poor filling of the vessels to the area of fibrosis in the left upper lobe. Five seconds after the dye had been introduced the left atrium was well filled, thus excluding a left atrial myxoma, but the pulmonary veins were abnormally distended (Fig. 4). The left ventricular wall was considered in retrospect to be unduly thick, though this feature was not appreciated at the time.

The patient was started on anticoagulants, in view of the possibility of multiple pulmonary emboli contributing to the pulmonary hypertension, and routine therapy of his cardiac failure was continued. He was also given antituberculous drugs. He showed further mild improvement and was discharged from hospital on 24 July 1959. Thereafter he was seen at monthly intervals as an out-patient. The clinical signs remained essentially unchanged with the raised venous pressure, distended liver and oedema persisting. Although seen by several experienced examiners, the auscultation findings of a tricuspid incompetent systolic murmur plus a gallop sound were all that were ever detected.

On 16 June 1960 the patient became extremely breathless and was again admitted to hospital. Examinations showed him to be in severe congestive cardiac failure with marked oedema of the extremities. The signs in the cardio-vascular system were the same as on previous occasions, but the gallop was now much louder and was present in all phases of respiration. He died on 23 July 1960.

PATHOLOGY

(*a*) *Autopsy Findings.* The autopsy was performed on 27 July 1960 at the Pneumoconiosis Research Unit, Johannesburg. Ankle and sacral oedema were noted. A marked bilateral, chronic, non-specific pleurisy with adhesions and a chronic anterior mediastinitis were present.

The heart weighed 640 g. and showed a chronic, non-specific, adhesive pericarditis. The weight of the left ventricular segment was 192 g. (*Normal*: 134-106 g.) and of the right 176 g. (*Normal*: 74-50 g.).

The aortic cusps were adherent, fibrosed and moderately stenosed (Fig. 5). There was adherence of 2 pulmonary cusps which were fibrosed at the site of adherence; the rest of the cusps were thin and pliable. (Fig. 6). Two smooth white nodules, each 4 mm. in diameter, were

present, one on the ventricular aspect of the tricuspid valve, and one on the auricular aspect of the posterior curtain of the mitral valve; both these valves showed no other lesion.

There was marked endocardial thickening in the left ventricle, most marked at the apex. The thickened endocardium measured 1.5 mm. in thickness (Fig. 7). Endocardial thickening to a less degree was present in the right ventricle, mainly affecting the upper half. No mixed ante-mortem thrombi were found.

Fig. 5. The aortic valve showing fibrosis, adherence of the cusps and stenosis.

Fig. 6. The pulmonary valve showing the adherence of two cusps.

The left auricle was markedly dilated and the ostia of the pulmonary veins were not stenosed. The myocardium, under the thickened endocardium, showed superficial fibrosis. The coronary arteries showed a slight degree of atheroma.

The lungs were congested and a well localized area of caseation 1½" in diameter was present in the left apical lobe. In addition a slight degree of silicosis was present.

The kidneys and liver showed a chronic venous congestion. The rest of the organs showed no gross change.

(*b*) *Histological Findings.* The pericardium was the seat of a chronic, non-specific pericarditis. The mural endocardium showed the features of primary endocardial disease in various stages of development. The earliest change was represented by a focal metachromasia with toluidene blue. In these areas a slight proliferation of reticulin and elastic fibres was present, the superficial fibres running parallel to the surface, the deeper fibres perpendicularly and extending into the underlying myocardium.

The endocardium in the region of the left ventricular apex showed a dense fibrosis with marked hyperplasia of the elastic tissue (Fig. 8). At the extreme apex, where the fibrosis was most marked, elastic tissue was absent and a small amount of calcium was present. Focal superficial deposits of fibrin were present on the fibrosed areas. The superficial parts of the fibrin deposits gave the normal staining reactions of fibrin, but the deeper portions stained a brownish-red colour with phosphotungstic acid haematoxylin.

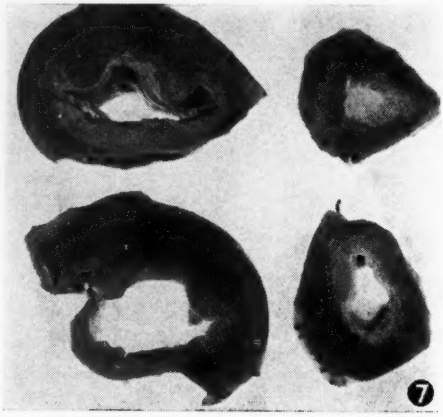

Fig. 7. Segments of the left ventricle showing the thickened mural endocardium.

The aortic valve was the seat of a fibroelastosis and showed focal deposits of fibrin in addition. These deposits were covered by swollen endothelial cells, some of which had penetrated into the fibrin mass and lay in lacunae (Fig. 9). No metachromasia was noted in the lacunae when stained with toluidene blue. The superficial portions of the fibrin deposits showed normal staining reactions but

the deeper portions stained a brownish-red colour with phosphotungstic acid and haematoxylin.

The pulmonary valve in the area of thickening showed a fibro-elastosis. The nodules on the mitral and tricuspid valves showed a dense fibrosis. The subendocardial myocardium showed vacuolation of the fibres, enlargement and hyperchromatism of the nuclei, focal necrosis and focal scarring. The rest of the myocardium showed no gross change. No lesions were noted in the vessels in the myocardium, but haemangiomatous foci were present in the deeper parts of the thickened endocardium (Fig. 10).

The lungs showed a well marked congestion but the pulmonary vessels appeared normal. The tuberculous focus in the left apical lobe appeared to be inactive and typical silicotic islets were present in the lungs. The kidneys and liver showed a chronic venous congestion.

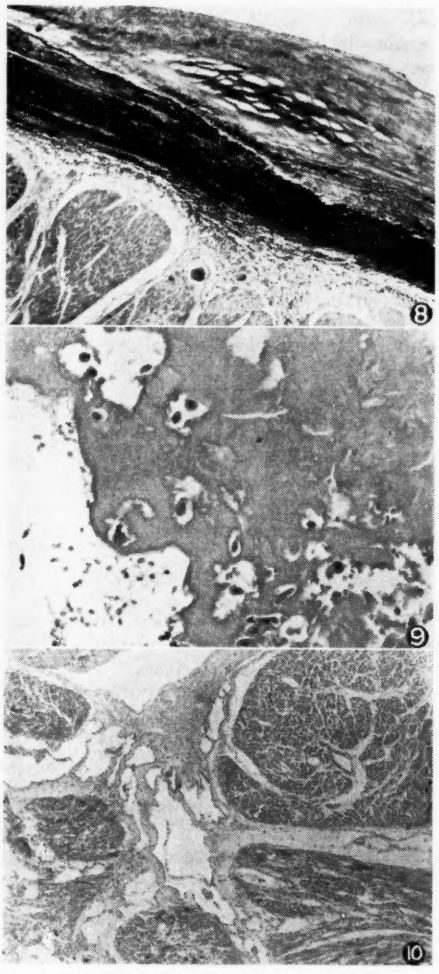

Fig. 8. A section of the mural endocardium of the left ventricle showing a fibro-elastosis. (× 4. Original magnification: × 10).

Fig. 9. A section of the fibrin deposit on the aortic valve showing the endocardial cells lying in lacunae in the fibrin mass. (× 26. Original magnification: × 64.)

Fig. 10. Section of the mural endocardium showing haemangiomatous foci in the fibrosed endocardium. (× 4. Original magnification: × 10.)

COMMENT

The clinical findings and the pathology of a case of the late chronic stage of primary mural endocardial disease are described. This type of primary mural endocardial disease is rare in this part of Southern Africa. The majority of cases seen here are in the acute, subacute and early chronic stage, never reaching the chronicity shown in this case.

In an attempt to dispel the confusion existing, it is suggested that the term primary mural endocardial disease be used to signify cases of idiopathic heart disease where the lesion appears to be endocardial in origin. The lesions in this disease are variable and what is found depends on the thoroughness of the examination, the number of histological sections examined and the stage of the disease. The disease is recurrent and usually acute, chronic and subacute lesions are found in the same case. The pericardium is not affected. The chronic, non-specific adhesive pericarditis in this case was almost surely due to tuberculosis.

During the course of this patient's illness it was realized that the pulmonary hypertension and right ventricular failure almost certainly resulted from, or were at least associated with, a left-sided lesion. Mitral stenosis was unlikely in view of the absence at all times of an apical mid-diastolic murmur and a left atrial myxoma was excluded by the angiocardiogram.[52] Prolonged left ventricular failure, such as could occur with ischaemic heart disease or one of the 'rare cardiomyopathies,'[53] including subendocardial fibrosis, seemed incompatible with the absence of clinical or radiological evidence of significant left ventricular dilatation. There were typical radiological signs of pleural thickening over the left lung and the postulation that fibrosis of the mediastinum by the

tuberculous process had resulted in partial obstruction of the pulmonary veins seemed a reasonable explanation of the patient's clinical picture. Pulmonary venous congestion, however, could clearly not explain the prominent left atrium on the X-rays or the conclusive evidence of left atrial enlargement on the electrocardiogram; these features, in retrospect, could only have resulted from chronic left ventricular failure in this case.

Although physical findings were against significant constrictive pericarditis, as was confirmed at autopsy, it is possible that the fibrosis of the pericardium may have prevented the degree of dilatation of the left ventricle which is usually seen in primary mural endocardial disease.[13] This was largely responsible for this very unusual clinical picture. Furthermore, the electrocardiographic signs of right ventricular strain without evidence of left ventricular hypertrophy were unlike those previously observed in this condition,[13] where RS-T segment and T wave abnormalities were the main features found. However, the case reported by Hudson *et al.*[5] also had had evidence of left atrial dilatation, pulmonary venous congestion, right ventricular hypertrophy and congestive heart failure, but necropsy showed a normal pericardium and extensive endocardial fibroelastosis of the left ventricle which, compared to the other 3 dilated chambers, appeared relatively contracted. Their patient had a clinical presentation similar to ours and although there had been no auscultatory signs of mitral stenosis, this diagnosis had been strongly suspected.

Either the rigid endocardium of the left ventricle or the mild degree of pericardial constriction might have accounted for the pulsus paradoxus. It is likely that the endocardial thickening, which was more extensive in the left ventricle, was the more important factor as no dip-and-plateau curve[51] was present in the right ventricle, yet the pericardium was equally thickened over both ventricles.

It is perhaps noteworthy that in spite of the appreciable pulmonary hypertension in our case (95/45 mm. Hg), the pulmonary vasculature showed little histological abnormality. This has been observed in several instances of severe pulmonary arterial hypertension due to left atrial myxomata.[48, 54] In those cases the pulmonary hypertension had been sufficiently severe to cause right ventricular failure and the haemodynamics were presumably similar to those present in this patient.

Although necropsy showed that the aortic valves of this case were fairly extensively involved by the pathological process, no aortic ejection systolic murmur had been heard and the absence of such a murmur is difficult to explain. It could admittedly be expected that the severe pulmonary hypertension and functional tricuspid incompetence would be associated with a small left ventricular stroke volume which might be the explanation. However, the systemic blood pressure was 130/70 mm. Hg, and it is surprising that the flow through the aortic valve was not sufficient to produce a basal systolic murmur radiating into the carotid arteries.

The 2 most probable causes of this condition are (*a*) auto-immune disease and (*b*) viral infection. In regard to an auto-immune disease, 2 factors support this view, viz. the absence of an inflammatory reaction in the lesions and the recurrences which occur. Mucoid oedema, fibrinoid deposits and eventual fibrosis are features of an auto-immune disease. The elastosis is a feature of the healing process in a constantly moving organ. There is nothing to support a viral or any other infection of the endocardium at the moment.

The enlargement of the heart which is frequently associated with this condition is interesting and difficult to explain but may be due to the subendocardial ischaemia.

SUMMARY AND CONCLUSIONS

1. A case is described in the late chronic stage of primary mural endocardial disease in a white gold miner aged 55 years.

2. The absence of inflammatory changes in this condition is striking and suggests that direct infection probably plays no part in the etiology.

3. An auto-immune disease with the endocardium as the target organ is suggested as the most probable cause.

4. Much research is needed into the etiology of primary mural endocardial disease.

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NOTES AND NEWS : BERIGTE

Dr. J. Graham Scott has been invited by World Health Organization to attend a session of the Expert Committee on Trachoma in Geneva in September 1961.

While overseas, Dr. Scott will also attend a meeting of the Jules Gonin Club to discuss modern methods of treating detachment of the retina. He will return early in October.

* * *

Dr. Leonard Schrire, M.B., Ch.B. (Cape Town), Dip. Clin. Path. (Rand), Dip. Bact. (Lond.), has joined Drs. K. G. Irving and D. Zent in practice as a pathologist at 1004 Metro Centre, Bree Street, Johannesburg. (*Telephones*:— *Laboratories*: 23-8579; *Residence*: 42-8404).

* * *

Dr. de Wet Vorster, M.B., Ch.B. (Cape Town), D.P.M. (England), has joined Dr. G. J. Goldberg in psychiatric practice at 404 Osler Chambers, Jeppe Street, Johannesburg. (*Telephone*:— Rooms: 23-9795).

HEALTH AND TUBERCULOSIS CONFERENCE, IBADAN, NIGERIA (MARCH 1962)

The Chest and Heart Association, with the co-operation of the Federal Government of Nigeria and by kind invitation of the University College, Ibadan, is sponsoring a Health and Tuberculosis Conference to be held at the University College, Ibadan, Nigeria from 26 to 31 March 1962.

This is a completely new venture and the aim is to encourage all workers in the field of tuberculosis in Africa. This is the sixth in the series of Commonwealth Conferences which have previously been held in London.

Among the speakers will be eminent chest physicians from Great Britain and all parts of the world. The programme will include lectures and panel discussions on:

Tuberculosis, the Greatest Tropical Menace;
Tuberculosis and Leprosy;
Community Infection—Cause and Remedy;
Drugs—Their Value and Limitations;
The Techniques of Community Surveys;
Non-Pulmonary Tuberculosis;

Travelling Clinics and Village Hygiene.

The Conference fee is 3 guineas and accommodation in the University hostels will be available at moderate charges. A charter plane will leave London on 24 March 1962.

Applications for further details should be made to:

The Conference Secretary,
The Chest and Heart Association,
Tavistock House, North Tavistock Square,
London, W.C.1 and *not* to the University College, Ibadan, Nigeria.

* * *

FRANK FORMAN MEDICAL FOUNDATION

1962 AWARD

The Board of Trustees of the Frank Forman Medical Foundation announce that:

1. The sum of Seven Hundred Pounds will be available for post-graduate award in January 1962.
2. In terms of the Trust Deed, the Board of Trustees are directed to use their discretion in making the Award, in such manner as shall promote or assist the study of medicine and/or medical research at the University of Cape Town or elsewhere.
3. The Award may take the form of:

A *Scholarship* to a medical graduate for post-graduate medical study for 1 year; and/or

A *Fellowship* to a university graduate for post-graduate medical research for 1 year; and/or

A *Grant,* either independently or in conjunction with other research grants, to any person, institution, or body, for special medical investigation or research.

4. Applications must be addressed to:

The Secretary,
Frank Forman Medical Foundation,
3, Park Road,
Rondebosch, Cape Province.

They must reach him before 30 November 1961.

* * *

AMERICAN COLLEGE OF SURGEONS

ANNUAL CLINICAL CONGRESS: CHICAGO

Latest applications of surgical research and new surgical techniques will be described at the world's largest meeting of surgeons, the *47th Annual Clinical Congress* of the American College of Surgeons, in Chicago 2-6 October, 1961.

More than 11,000 surgeons and physicians, from all over the United States, Canada and many foreign countries are expected to attend this widely instructive 5-day meeting. About 1,000 doctors will take part in the programme of 9 postgraduate courses, 258 new research reports from medical centers throughout the country, 68 medical motion pictures, 26 cine clinics, 14 operative telecasts from Billings Hospital of the University of Chicago, and 300 scientific and industrial exhibits.

Dr. John T. Reynolds, Chicago, Clinical Professor of Surgery, University of Illinois College of Medicine, is Chairman of the Committee on Local Arrangements.

Major addresses will be made by Dr. Robert M. Zollinger, Ohio State University College of Medicine and incoming president of the College, speaking on *Surgical Tithing*; Dr. Francis D. Moore, Harvard Medical School, giving the annual Baxter Lecture on *The Control of Effective Volume and Tonicity: Body Composition,* and Dr. Preston A. Wade, Cornell University Medical School, presenting the annual Trauma Oration, *The Specialist and the Injured Patient.*

This year's historic Martin Memorial Lecture, commemorating the College founder, Dr. Franklin H. Martin, will be given by Admiral Hyman G. Rickover.

Honorary Fellowships will be awarded to 4 distinguished surgeons: Sir Arthur Porritt, London, President of the Royal College of Surgeons of England; Dr. Jean-Louis Lortat-Jacob, Professor of Surgery, University of Paris; Dr. Fritz Linder, Professor of Surgery, Free University of Berlin; Dr. Jack F. E. Adams-Ray, Professor of Surgery, Karolinska Institute, Stockholm. Two Distinguished Service Awards will be given and more than 1,050 surgeons will become Fellows of the College (F.A.C.S.) at convocation ceremonies.

Nine postgraduate courses will be headed by surgeon-teachers prominent in their fields:

Pre- and Postoperative Care, James D. Hardy, Jackson, Miss.;

Gastrointestinal Disease, Stanley O. Hoerr, Cleveland;

Diseases of Liver, Biliary, Tract, Pancreas, Ben Eiseman, Louisville;

Cardiovascular Surgery, Ivan W. Brown, Durham:

Injury of Joints of Upper Extremity, Sam W. Banks, Chicago;

Obstetrics and Gynecology, David Danforth, Evanston, Ill.;

Cancer Chemotherapy, Harry S. Morton, Montreal;

Thoracic Surgery, Hiram T. Langston, Chicago;

Recent Advances in Pediatric Surgery, H. William Clatworthy, Jr., Columbus.

Dr. William P. Longmire, Jr., Los Angeles, presides over a session on *What's New in Surgery,* assisted by authorities in each speciality:

Neurological Surgery, William H. Sweet, Boston;

Gastrointestinal Biliary Conditions, Edward R. Woodward, Gainesville, Fla.;

Plastic Surgery, Truman G. Blocker, Jr., Galveston;

Tumors, William D. Holden, Cleveland;

Anesthesia and Pulmonary Problems, William S. Blakemore, Philadelphia;

Cardiovascular Surgery, C. Rollins Hanlon, St. Louis;

Orthopedic Surgery, William Stanley Smith, Columbus, Ohio.

Dr. Harrison L. McLaughlin, New York, will lead a *Trauma* symposium covering *Tetanus Prophylaxis, Present Status of Plastic Polymers in Fracture Treatment, Prevention and Care of Athletic Injuries,* and *Causes of Death Following Trauma.* Dr. R. Lee Clark, Houston, will lead a cancer panel on *Alterations in the Hormonal Balance of the Cancer Patient.*

Thyroid Disease, Emergency Department Problems in Trauma, Facial Injuries, Skin Grafts, Head Injuries, Viral Infections in Pregnancy, Knee Joint Fractures, Duodenal Ulcer, Diseases of the Spleen, Cerebral Vascular Insufficiency, and *Tumors of the Kidneys* are additional surgical problems to be discussed by specialists.

Dr. I. S. Ravdin, Philadelphia, is retiring president of the College. Dr. John Paul North, Chicago, is the Director. Headquarters for this meeting is The Conrad Hilton, with some sessions at nearby Loop hotels.

'IRON LUNG' DEVELOPMENT

Since 1954 Associated Electrical Industries Ltd. has been the exclusive supplier of fractional horsepower electric motors for use with iron lungs made by the Cape Engineering Company Ltd. at Warwick, England.

The Kelleher Rotary Senior Respirator (a so-called iron lung) was shown at the International Hospital Equipment Exhibition at Olympia in May 1961. It is a development of the well-known Smith-Clarke 'Aligator' Respirator. Although the mechanical breathing characteristics are the same, there are 2 important differences:

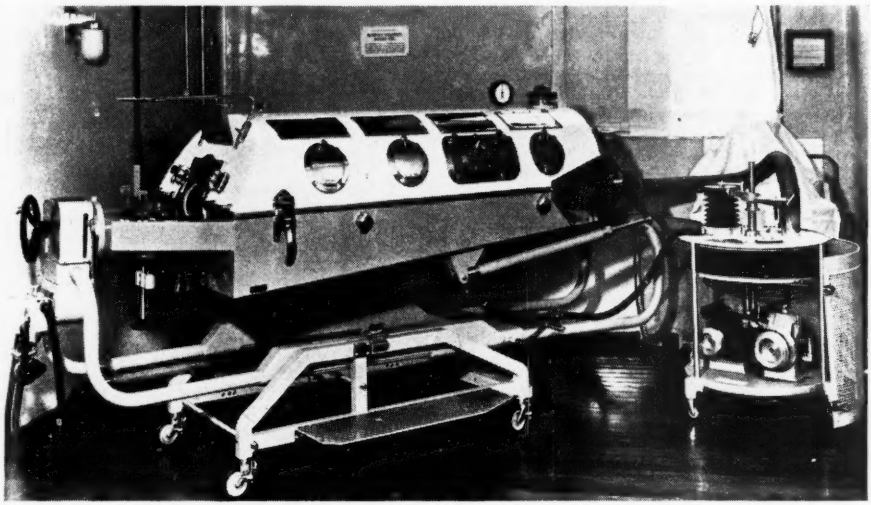
1. Without interrupting breathing the patient can be turned from the supine to the prone position, or anywhere in between, by one person. To achieve this the cabinet in which the patient lies can be turned through 180° either left or right of the normal horizontal position; but to simplify and safeguard the electrical connexions it can only be returned through the arc described.

The cabinet itself has a triangular frame extension at either end, each extension terminating in a trunnion bearing supported in one of the raised ends of a long U-shaped tubular frame work.

2. Once in the prone position, the medical attendant has manual access to the patient's back, again without interruption of breathing.

The patient is enclosed from the neck downwards, and access by the medical attendant is through the airtight armhole ports on the side of the cabinet.

The breathing apparatus is operated by means of an AEI $\frac{1}{6}$ horsepower electric motor—normally of a standard type—operating on a 230 volt a.c. 50 local needs. The motor drives a gearbox through a cycle supply. The design can be modified to meet



General view of respirator showing pump unit (on right) with AEI fractional h.p. motor

5 step pulley, the gearbox being connected to a crank which drives a rod which in turn operates 2 sets of bellows, one large and one small. The large bellows is connected by a tube to the inside of the cabinet and breathing is achieved through a raising and lowering of the atmospheric pressure. By means of the 5 speed pulleys breathing rates can be varied from 16 to 32 per minute.

The cabinet, or respirator, is of course normally airtight but when for various reasons this airtight condition has to be interrupted, the patient is enabled to breathe by means of a face mask which is operated by the small bellows. In this case air is forced into and drawn out of the lungs.

The action of both sets of bellows is mechanically synchronized so that, when medically desirable, there are 2 means of assisting the patient.

REVIEWS OF BOOKS

THE MERCK MANUAL

A NEW, 10TH EDITION

A revised, expanded and updated tenth edition of the *Merck Manual* in English was published in June of this year by the Merck Sharp & Dohme Research Laboratories Division of Merck & Co., Inc., of Rahway, New Jersey, as part of a programme to serve the medical and allied professions.

The 1,900 page, thumb-indexed book comprises 384 chapters on the diagnosis and treatment of diseases, categorized into 21 main sections each covering a specific field of practice.

The *Merck Manual*, first published 60 years ago as a service to the medical and allied professions, has gained worldwide repute as a reference work

providing physicians with well organized, up-to-date facts, facilitating accurate diagnoses and promoting effective treatment. Characterized as a doctor's bible, it has been termed a 'must' for every medical library and is actively used in medical schools and for post-graduate studies.

More than 100 authorities in various fields of medicine served as authors or consultants in preparing the new tenth edition of the *Merck Manual*.

Several hundred carefully selected prescriptions are included embodying the most updated advances in medicine. Along with 9 special therapy chapters they are conveniently grouped in one section and categorized according to clinical indications.

The tenth edition of the *Merck Manual* includes 20 new subjects, many original illustrations and numerous new Tables. Thus the tenth edition has kept pace with the progress in modern medicine.

The coverage of a wide variety of disorders such as disturbances in inorganic metabolism, dental and oral defects plus the malabsorption syndrome, group A streptococcal infections, toxoplasmosis, pulmonary granulomatoses, and genetic metabolic anomalies has been broadened, while sections dealing with the care of normal newborns and preschool children; antihistamine, thrombolytic, diuretic and psychopharmocologic therapy; the dermatitides; resuscitation methods; tubeless gastric analysis, rheumatoid arthritis tests, identification of tumour cells in body fluids, and the diagnostic use of radioisotopes, have been brought up to date.

A special section of the Manual provides a 'how-to-do-it' on clinical procedures, nursing techniques, office laboratory and selected paediatric and immunization methods. In this section pre-operative and post-operative routines, dietary exchanges and other feeding directions as well as practical reference Tables are instantly available.

The tenth edition is printed in pocket-size on strong Bible paper in the traditional dark blue, gold stamped durable binding.

The Regular Edition is priced at US $7.50, while a De Luxe Edition is also available at US $9.75 with gold edged pages. Physicians and members of allied professions may order directly from the Publication Department, Merck & Co., Inc., Rahway, New Jersey, U.S.A.

KINESIOLOGY

Kranz' Manual of Kinesiology. Clem W. Thompson, Ph.D., F.A.C.S.M. (1961. Pp. 159 + Index. With Illustrations. R 3.20).
St. Louis: The C. V. Mosby Company. Obtainable from local booksellers.

This is a manual designed to provide a teaching textbook for teachers in Physical Education. There are 11 chapters, dealing specially with joints and their movements and their skeletal and muscular systems. Numerous anatomical drawings illustrate the bones and muscles together with schematic representations of joint action and muscle movement.

A number of drawings at the end of the manual show the anterior and posterior aspects of the human skeleton. These pages are perforated and are intended for practical use in teaching.

The anatomy is sketchy and, while the manual may meet the requirements for physical educators, if used by the student in anatomy it must be used in conjunction with the standard anatomy textbooks.

However, to the student in kinesiology in Physical Education, the manual does what it sets out to do. It provides a very brief, organized manual on the skeletal and muscular systems for a particular type of student.

HEREDITARY CHONDRODYSPLASIAS

Problems of Hereditary Chondrodysplasias. By Andreas Hobaek. (1961. Pp. 175. With 49 Plates. R 5.40).
Oslo: Oslo University Press.

The hereditary chondrodysplasias form a complex group of lesions. Some cases fall readily into named groups, but many that are incomplete or mixed forms are difficult or impossible to classify. Dr. Hobaek presents a new classification:

1. Gargoylism.
2. Chondrodystrophia foetalis (achondroplasia).
3. Chondrodystrophia calcificans congenita.
4. Chondrodysplasia hereditaria infantilis:
(*a*) General Lesions:
i. With extracartilaginar lesions.
ii. Without extracartilaginar lesions.
(*b*) Mainly epiphyseal.
(*c*) Mainly axial (vertebral).
5. Unclassifiable.

This has the merits the author claims: '. . . it affords possibilities for necessary differentiation, it is fairly simple, and it allows registration of the large majority of cases . . .' It also has the weakness inherent in any classification that must, perforce, ignore etiology and pathogenesis, but the whole concept is stimulating and it may be a stepping stone to real understanding.

The book describes the clinical and radiological features of a large number of cases in many families, and most of the X-ray reproductions are passably good. Many, however, are poor and not all are useful. Some can be correlated with the text only with distracting difficulty. Much of the descriptive terminology lacks definition. Striking abnormalities, in one case even a dislocated hip, may escape mention. 'Hyperthyroidism' is used in place of 'hypothyroidism' with irritating frequency. The result is confusion no less confounded. Nevertheless this book is well worth reading for its basic unitarian concept and for its wealth of material. With competent medical editorial advice it could have been a classic contribution.

FRACTURES, DISLOCATIONS AND SPRAINS

Key and Conwell's Management of Fractures, Dislocations and Sprains. H. Earle Conwell, M.D., F.A.C.S. and Fred C. Reynolds, M.D. (1961. Pp. 1129 + Index. With 1227 Illustrations. R22.95).
St. Louis: The C. V. Mosby Company. Obtainable from local booksellers.

The immediate reaction to the seventh edition of Key and Conwell's *Management of Fractures, Dislocations and Sprains* is similar to that experienced when, after a lapse of years, we meet an old friend. Soundly conservative, but somewhat changed and improved, mature and informed on modern trends, immaculately turned out, but still pre-eminently a worthy friend.

The old format, with *Principles and General Aspects* followed by the *Treatment of Specific In-*

juries, is continued and in this latter section the résumé of the anatomy, at the beginning of each chapter, of the particular region to be dealt with, is practical and makes for easy reference.

In the section on *Spine Injuries*, the division of fractures into stable and unstable varieties, with the difference in treatment and the reasons for this is clearcut. The ever-increasing whiplash injuries to the cervical spine are also dealt with. The more recent work from Sheffield, on fractured spines, especially with paraplegia, is not included.

In extremity fractures increasing use of metallic fixatives, particularly intra-medullary nails, is noted; but generally there is still a healthy conservatism in the treatment of fractures. The indications and contra-indications to intra-medullary fixation are dealt with. The method of securing stability with multiple Kirschner wires, thereby allowing earlier movement in the treatment of hand injuries, promises the possibility of better results in this type of surgery.

Under the head of *Strains* numerous conditions are dealt with in their respective sections. These include such topics as *Intervertebral Disc Injuries, Rupture of the Meniscae and Ligaments in the Knee, Rotator Cuff Injuries about the Shoulder Region* and even *Foot Strain*.

It might be expected that in attempting to encompass such a wealth of topics, as well as the infinite varieties of fractures and dislocations, some might receive inadequate description; but, with few exceptions, a good balance is maintained throughout, bearing in mind that conservative treatment of the average fracture can be described more briefly than operative treatment for those more complicated fractures which warrant this latter treatment.

One confusing aspect to readers outside America is eponymous usage to denote specific forms of treatment, especially where the name is not associated with the particular treatment in other countries. Cross-reference, however, serves to clarify these points.

On the whole this book should prove a useful addition to the libraries of those not infrequently called upon to deal with fractures. dislocations and sprains.

THE CATARRHAL CHILD

The Catarrhal Child. By John Fry, M.D. (Lond.), F.R.C.S. (Eng.). (1961. Pp. 131 + Index. With Figs. R 2.50 + Postage 10c).

London and Durban: Butterworth and Co. (Publishers) Limited.

The catarrhal child is a great source of worry and anxiety both to his family and to his general practitioner. The problem is an extremely common one. This monograph gives us an excellent clinical picture of this difficulty. The authors discuss all the aspects from the medical, psychological and practical points of view. One is particularly impressed by the clinical approach and deduction based on accurate facts and figures.

The problems discussed include coughs and colds, acute chest infection, the wheezy child, tonsils and adenoids and the use of antibiotics. The latter chapter is particularly valuable. Indications for their use are clearly defined, the clinical state, height of fever and age of patient being the determining factors. The liberal use of Aspirin and symptomatic measures, e.g. antispasmodics in asthmatic attacks, should replace the all too frequent prescription for antibiotics. Tonsillectomy is viewed in its correct perspective and a plea is made for research into its indications, since in Great Britain over the past 10 years 2 million children have had their tonsils removed at a cost of over £32 million and a loss of 350 lives.

The natural history of many diseases, e.g. peptic ulcer, hypertension, chronic pyelonephritis, etc. is best studied in the patients' natural environment—his home. The family doctor can make significant contributions to clinical medicine if he follows the example of Drs. Fry and Pickles.

PHARMACEUTICAL ADVERTISING

Pharmaceutical Advertising: A Survey of Existing Legislation. Offprint from *International Digest of Health Legislation*, 1961, vol. 12, No. 1, 53 pages, Geneva. R 0.35.

Pretoria: Van Schaik's Bookstore (Pty.), Ltd., P.O. Box 724.

Throughout the world the number of pharmaceutical specialities is rapidly increasing, and there is a corresponding increase in pharmaceutical advertising. Intensive propaganda is liable to expose the general public to various risks, including the dangers of self-medication. Legislation laying down strict standards is thus essential in order to safeguard public health and obviate the making of extravagant claims. as well as to ensure that the public is neither caused unnecessary alarm nor induced to delay seeking treatment when this is needed. Although a number of countries still have no legislation on pharmaceutical advertising, or are drafting or revising their regulations, some have enacted provisions dealing with this subject, especially since 1940.

The comparative study just published in the *International Digest of Health Legislation* gives an analysis of the existing legislation in some 22 countries.

OBSTETRICS AND GYNAECOLOGY

Contributions to Obstetrics and Gynaecology. V. N. Shirodkar, M.D. (Bombay), F.R.C.S. (Eng.), F.A.C.S. (1960. Pp. 150 + Index. With 196 Figs. R 3.75 net. Abroad Postage 17½c).

Edinburgh and London: E. & S. Livingtone Ltd.

This work was prompted by the author's inability. as he himself says, to reconcile himself to some of the older standard operative procedures. It is on this account stimulating and refreshing, particularly for the reader who has become weary of repeated plagiarism in textbook writing, to examine radical departures in operative technique, even if he does not agree with all that is said.

Outside his own country Professor Shirodkar is probably best known for his operative treatment of habitual abortion in the second trimester. The aetiology of cervical incompetence is fully discussed here and the surgical treatment extremely well illustrated in the first chapter. This aspect of Shirodkar's work certainly deserves the serious consideration of every gynaecologist.

The author deals at much greater length with the problem of uterine prolapse and describes his

extended Manchester operation and the intraperitoneal sling procedure. Compared with the Fothergill operation and with vaginal hysterectomy, his revised techniques are extremely radical and complicated and, whether they are justified as routine treatment, must be left to the judgment of the individual surgeon, according to his skill and experience.

Many excellent photographic illustrations are contained in the sections dealing with the surgery of blocked Fallopian tubes and the construction of an artificial vagina.

This work concludes with a comprehensive summary of various papers by the author, who is a surgeon and teacher of vast experience and who for 20 years (up to 1955) held the Chair of Obstetrics and Gynaecology at Grant Medical College, Bombay.

A SOUTH AFRICAN MEDICAL WHO'S WHO

The Medical Directory of South Africa. Ed. by B. Crowhurst-Archer, M.D. 1960. Pp. 360. Durban: The Knox Printing Co. (Pty.) Limited.

This is the first edition of a South African *Medical Who's Who*. It has been produced under the enterprising and enthusiastic editorship of Dr. B. Crowhurst-Archer, of Durban, and apparently with the approval of the South African Medical Council.

It lists the names of all practitioners who appear in the official Register of Medical Practitioners issued by the South African Medical Council, but an attempt has been made to provide some biographical details (including titles of papers published) where this information has been made available.

There is also a considerable amount of collateral medical information, including the very useful list of medical periodicals available in the libraries of the medical schools in South Africa, as well as a list of Institutes for Alcoholics and an Index to Government and Provincial Hospitals.

It is unfortunate that, in a publication released in 1961, *Geneeskunde* (the only Afrikaans medical journal in the country) and *Medical Proceedings*, have both been omitted, as well as other important periodical medical publications. Such omissions will no doubt be rectified in future editions.

The progressive and continued success of this Medical Directory depends upon the co-operation of registered medical practitioners in supplying biographical and other information.

MODERN TRENDS IN CARDIOLOGY

Modern Trends in Cardiology. Ed. by A. Morgan Jones, M.Sc., M.B., F.R.C.P. (1960. Pp. 264 + Index. With 42 Figs. R 7.00 plus 25c postage).

London and Durban: Butterworth & Co. (Publishers) Ltd.

This book is the most recent addition to the *Modern Trends* volumes, and is ably edited by A. Morgan Jones. It deals with many aspects not to be found in textbooks, even recent ones, and should prove useful to the cardiologist and the specialist physician. No less than 6 of the 16 articles are concerned with coronary artery disease, and reflect the current interest in this important subject.

Like all books with articles contributed by different authors, the subject matter is uneven. Butler's article on *Pulmonary Function in Heart Disease*, while valuable to the enlightened, is too obscure for those not conversant with pulmonary function tests. *The Measurement of Coronary Flow in Health and Disease*, by Richard Gorlin, illustrates well the difficulties of measuring the coronary flow in man, as well as dealing with the practical aspects of coronary flow and is a very suitable article for a book of this type. Some articles are mainly clinical, such as *Medical Aspects of Cardiac Surgery*, by Campbell, and *Atrial Septal Defect*, by Evan Bedford and Holmes Sellors

The Management of Congestive Heart Failure, by Oscar Brenner, concerns itself mainly with the author's personal views and experience, and is highly original.

D. A. K. Black has contributed a valuable article on *Electrolyte Metabolism in Heart Disease* and, as usual with this writer, has made a difficult subject appear relatively easy.

The articles by Epstein on *Epidemiology of Coronary Heart Disease* and Oliver on *Metabolic Factors in the Aetiology of Coronary Heart Disease* show the complexities of this subject, and confound those who believe in a purely dietary aetiology for coronary heart disease.

Notable omissions in this book are references to modern methods of cardiac investigation, especially cardiac catheterization, angiocardiography (and in particular cine-angiocardiography) and, most important of all, dye-dilution studies, all of which methods are of established importance in cardiac diagnosis. It is hoped that these omissions will be made good in future series.

The book is well printed on art paper, and should be a welcome addition to any library.

ARTHROPOD-BORNE VIRUSES

Study Group on Arthropod-borne Viruses: Report. World Health Organization: Technical Report Series, 1961, No. 219; 68 pages. R 0.50.

Pretoria: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724.

The continual discovery of new arthropod-borne viruses causing disease in man and animals is of great significance in public health and in veterinary medicine, a full assessment of their importance depending upon the development of improved diagnostic and reporting systems.

This *Report* discusses the promotion and co-ordination of research into these viruses. It outlines the procedures for determining their presence in hitherto unexplored or incompletely studied areas, and deals with the characteristics of enzootic foci, serological surveys (serological techniques, sampling methods, the preservation of serum specimens, etc.), and virus isolation (materials and methods, procedures for examining specimens, and identification of recovered viruses).

Classification systems, the criterea for acceptance of an arthropod-borne virus, and serological grouping and identification are examined. Attention is drawn to those fields of research where increased knowledge is essential for progress towards a better system of classification. The clinical, epidemiological, geographical and virological criterea for the diagnosis of virus infections are reviewed, and techniques of

case-finding and systems of reporting discussed.

Other subjects covered include ecological aspects —biological and physical factors that influence virus transmission; the establishment and functions of reference laboratories, both central and regional; information exchange procedures, including an *Arthropod-Borne Virus Information Exchange* Newsletter and a catalogue of arthropod-borne viruses. The *Report* concludes with an examination of control measures, summarizing the present position with regard to vaccine development, and giving illustrations of some important methods of vector and host control.

Appended to the *Report* is a series of *Annexures* tabulating natural sources and isolation and identification of arthropod-borne viruses; antigenic groupings; human infections caused by these viruses; confirmed arthropod-borne virus diseases to be reported to WHO: FAO as sporadic cases or epidemic outbreaks; the data to be provided on submission of virus strains for identification; and the names and addresses of ornithological centres specially devoted to miscellaneous collecting and taxonomic identification and of some outstanding parasitological centres for the biological study and identification of ectoparasites. Also included are facsimiles of a questionnaire for collecting information on virus strains, a punch card presenting data on an arthropod-borne virus, a key to the punch card catalogue of viruses, and the text of a letter to prospective contributors to the catalogue.

CARCINOGENICITY OF FOOD ADDITIVES

Evaluation of the Carcinogenic Hazards of Food Additives: Fifth Report of the Joint FAO: WHO Expert Committee on Food Additives. World Health Organization: Technical Report Series, 1961, No. 220, 36 pages. R 0.35.

Pretoria: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724.

Chemical substances have been added to foodstuffs since time immemorial, to improve their appearance or flavour, to enhance their keeping quality or stability, or to assist in their processing. These additives have often been used without a proper investigation into their effect on health and in recent years some of them have been suspected of causing cancer.

This *Report* of a Joint FAO: WHO Expert Committee surveys the dangers of cancer from food additives. Information, the Committee points out, is lacking on the toxicity or potential carcinogenicity of many food additives, and a large number of the tests for carcinogenicity, on which the evidence for or against their harmlessness is based, are inadequately designed and executed, the results achieved not properly interpreted. The Committee accordingly examines in detail the principles to which such tests should conform, the factors that must be taken into account, and the criteria on which the interpretation of the results must be founded. It is particularly difficult in cancer—the report states—to apply to man the results of experiments in animals, and as yet not enough is known about the possible summating or potentiating effects of the different carcinogens in the human environment. While the ideal is to exclude all carcinogenic agents from foodstuffs, this is not, for a variety of reasons, always possible, and the risk of the additives must be carefully balanced against their benefit to the community and the individual consumer.

The *Report* indicates the fields in which more research is needed. One of the two annexes contains information on the carcinogenicity of food additives and contaminants examined by the Committee, the other the data that in the Committee's view should be contained in publications reporting investigations on carcinogenicity.

REHABILITATION IN LEPROSY

Scientific Meeting on Rehabilitation in Leprosy: Report. World Health Organization: Technical Report Series, 1961, No. 221; 37 pages. R 0.35.

Pretoria: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724.

Of an estimated world total of 10 million people suffering from leprosy, probably not more than 20% are receiving treatment of any kind for the severe disabilities that the disease often causes. The Scientific Meeting on Rehabilitation in Leprosy, through an on-the-spot examination in leprosy patients with typical varieties of deformity at a research sanatorium in India, set out to determine the extent of the problem of disability, to advise on the best application of available knowledge in prevention, treatment, and rehabilitation of disabled leprosy patients, and to recommend further research.

The *Report* of the meeting classifies the conditions which produce deformities under the following headings: nerve involvement, trophic ulceration, bone changes and absorption, deformities of the face, and ocular damage. Each condition, and the disablement it produces, are described in detail; suitable treatment—medical, surgical, and physiotherapeutic—is recommended; and suggestions are made for research into some hitherto unsolved problems. A classification of deformity devised by the WHO Expert Committee on Leprosy is presented in an *Annexure.*

The *Report* attaches great significance to the restoration of treated leprosy patients to normal physical and mental activity, through rehabilitation, reconstructive surgery, and re-education. It describes methods of combating some of the special difficulties inherent in their social reintegration—for example, by educational campaigns and re-employment schemes.

The training of health personnel and social workers is singled out as being the greatest requirement for efficient leprosy control in areas with limited health services. The importance of auxiliary workers is emphasized, and the kind of training needed to enable them to recognize and prevent deformities at an early stage is described in an *Annexure.*

MATERNAL AND CHILD HEALTH

Expert Committee on Maternal and Child Health (Third Report): Public Health Aspects of Low Birth Weight. World Health Organization: Technical Report Series, 1961, No. 217; 16 pages. R0.17½.

Pretoria: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724.

A decade ago it was realized that the existing international definition of prematurity (a birth weight of 2,500 g. or less) adopted by the World Health Assembly in 1948 would not be applicable in certain countries. While it is of value as a criterion for distinguishing infants requiring special attention

(and hence is useful in planning public health programmes), adherence to it as a standard has resulted in the occasional reporting of unusually high percentages of premature babies. It was observed that a large number of these infants, for whom special care was often not available, nevertheless grew very well. Lower birth weight levels were often adopted locally as the standard for prematurity. This resulted in confusion and made comparisons with other areas extremely difficult. The WHO Expert Committee on Maternal and Child Health therefore judged the time suitable to reassess the situation, on the basis of the preliminary results (covering 23,000 births) of a Study on Birth Weight carried out in 37 institutions in 18 countries and of working documents specially prepared by 22 experts in this field from various parts of the world.

The principal findings of the Committee concern the part played by environmental factors in successful child-bearing. A relationship between low birth weight and poor environment has been firmly established, but the basic mechanisms involved are not yet well understood and much further investigation is needed. Assuming, however, that the vast majority of newborn infants have the potential for being born with a birth weight consistent with full viability and health (dependent on an expected general improvement in the environment of the maternal population), the Committee decided that it would be inappropriate now to propose changing the upper limit of prematurity in the international definition. They recommend, therefore, that the concept of 'prematurity' in the definition should give way to that of 'low birth weight.' They further propose that, to meet the need for more facts, birth registration should be as complete as possible, and that, as soon as is practicable, birth weight be added to the official birth certificate used in each country. Recommendations are also made on methods for collecting information, on the preventive aspects of a programme for babies of low weight, on the care of these infants (with particular emphasis on special care by simple means), on the educational aspects, and on needed research.

HEALTH STATISTICS

Expert Committee on Health Statistics: Seventh Report. World Health Organization: Technical Report Series, 1961, No. 218; 28 pages. R 0.17½.

Pretoria: Van Schaik's Bookstore (Pty.) Ltd., P.O. Box 724.

The WHO Expert Committee on Health Statistics in its latest report discusses the supplementing of existing health statistics by health survey programmes based on modern scientific survey procedures adapted as appropriate to different regions. The characteristics of each kind of survey are distinguished, and advice is given on selecting the most suitable for varying objectives and circumstances.

The Committee reviewed the report of the UN/WHO Seminar on the Use of Vital and Health Statistics for Genetic and Radiation Studies (1960) and recommended that countries should take cognizance of the suggestions set out in the "Consensus of Opinion of the Experts participating in the Seminar" (reproduced as an annex to the present report). The Committee also emphasized the need for further development of hospital statistics from an international point of view. Other items discussed include the WHO statistical programme, and activities of national committees on vital and health statistics.

CORRESPONDENCE

DURBAN PROVINCIAL HOSPITALS AS COMPETITORS OF PRIVATE MEDICAL PRACTICE

CRITERIA FOR RECOGNIZING MEDICAL AID SOCIETIES

To the Editor: In a circular dated 3 July 1961 to members of the Durban Municipal Employees' Medical Aid Society, their Honorary Secretary-Treasurer writes:

'*Hospitals and Nursing Homes.* The benefit of accommodation in *private Nursing Homes* has been reduced by R 0.50 a day but increased by R 0.50 a day for *Provincial Hospital* patients. The accommodation fees for patients at Addington and Wentworth will therefore be paid in full by the Society plus R 8.40 towards the cost of diagnostic X-rays.'

I was formerly under the impression that the principle underlying the existence of Approved Medical Aid Societies was to enable lower and middle income families to enjoy the benefits of private medical treatment! In Durban all the Provincial Hospitals are *closed* hospitals. There are no private patients. No hospital patient has the right to be treated by his or her private doctor at these hospitals. The majority of the doctors on the hospital staffs are whole-time salaried medical officers, and *no* hospital doctors (not even the part-time visiting doctors) render any accounts to hospital patients. The hospital provincial accountants render all the accounts to hospital patients, and the province keeps all the resulting fees paid by everyone and every authority.

It is quite clear therefore that the Secretary-Treasurer of the Durban Municipal Employees' Medical Aid Society prefers his members to be treated by *Durban Provincial Hospitals* which are the *major competitors of private medical practice* in Durban.

It is time therefore that the Medical Association of South Africa scrutinized *all* the rules and regulations of *each* approved Medical Aid Society individually to see whether their policies really do enable lower and middle income groups to enjoy the benefits of private medical treatment, or *not*.

It is also time for the Durban Provincial Hospital authorities to take stock of the extent to which *they* are strangling private medical practice by allowing Medical Aid Society patients to be admitted to the precious hospital beds which should be reserved for *emergencies,* specially selected cases and the *indigent.*

'Private Practice.

Durban.